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VIRUS AND VIRUSLIKE DISEASES OF CITRUS

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Virus diseases . . .

are a threat to California's citrus industry. They are less readily identifiable than some other types of plant diseases, and treatment for affected trees consists largely of preventive rather than curative measures.

This circular . . .

lists the major virus and viruslike citrus diseases, based on recent research by workers in citrus-growing areas around the world. It describes symptoms and indicates possible control measures. Illustrations are included, for help in identification, and a table is also provided for easy reference to specific diseases.

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VIRUS AND VIRUSLIKE DISEASES OF CITRUS

Diseases caused by viruses and mycoplasmalike bodies are among the greatest threats to citrus. Viruses consist of either ribonucleic acid (RNA) or deoxyribonucleic acid (DNA), usually surrounded by a very thin protein coat. Mycoplasmas resemble small "L" (irregularly shaped) forms of certain bacteria, and are thousands of times larger than viruses. Mycoplasmas contain both RNA and DNA, and have a complex internal structure, including ribosomes, all enclosed within a membrane. Mycoplasmas reproduce themselves, whereas virus replication is an activity of the infected host under the influence of the virus. Viruses are incapable of reproducing themselves *in vitro*. Recent evidence suggests that stubborn disease (*34*)* and greening disease (*41*), long considered to be caused by viruses, may instead be caused by mycoplasmalike bodies. Mycoplasmalike diseases and virus diseases are graft-transmissible. (Certain other diseases with viruslike symptoms, but apparently not transmissible by grafting, are discussed at the end of the text.)

The citrus industry is being greatly benefited by international cooperation in exchange of information on the culture and diseases of citrus. In 1957, the International Organization of Citrus Virologists was formed at Riverside, California. Since then, the group has held its conferences in several of the world's citrus-growing areas, and has published its proceedings and accounts of current research (*23, 52, 53, 54, 74*). The first conference, in 1957, was held in California. Following ones have been held in Florida, Brazil, the Mediterranean countries of Italy, France, Spain, and Morocco, and in Japan, Taiwan, and the Philippines.

The control of virus and mycoplasmalike diseases of citrus is achieved principally by the planting of trees that are disease-free and either tolerant of or rarely susceptible to infection under field conditions. While occasional transmission through seed has been noted for some virus diseases, the use of nucellar seedlings is still an important means of freeing a desirable citrus variety from destructive viruses. Heat therapy, tissue culture, and chemicals are also useful in obtaining propagative stock free of graft-transmissible diseases. Citrus registration and certification programs, pioneered in California and designed to secure the best disease-free propagative materials, are being put into effect in much of the citrus-growing world. Citrus tree registrations and certifications in California are processed by the Nursery Service of the California Department of Agriculture, Sacramento.

VIRUS DISEASES

PSOROSIS

Psorosis, also called "scaly bark" in California, is worldwide in distribution. It should not be confused with leprosis, which is called "scaly bark" in Florida (*23, 70*), where psorosis also occurs. The different forms of psorosis and their possible relationships have been discussed elsewhere (*20, 23, 30, 31, 36, 73, 74*). Psorosis virus causes symptoms on leaves as well as on the bark. On leaves, many small, elongated, light-colored areas occur in the region of the small veinlets (fig. 1). On young, tender leaves not only the

* Numbers in parentheses refer to "Literature Cited," in which many general publications on citrus virus diseases and some comprehensive or special articles on certain diseases are listed.

Fig. 1. Psorosis symptoms (upper) on sweet orange leaves; (lower) on lemon leaves.

veinlets themselves, but also adjacent tissue may show faint to pronounced clearing. These small, cleared places, about 1 to 3 mm long and ¼ to 1 mm broad, may be numerously scattered over the entire blade, or may occupy only certain portions. Sometimes only a small percentage of young leaves shows these symptoms, but at other times a majority will show them. Often the pattern is not distinct; spots either fade out gradually at the margins or run together. Under hot summer conditions the flecks fade and are difficult or impossible to detect.

In some specimens only the clearings along the veinlets are apparent. To see these symptoms most readily, observe young fourth- to half-grown leaves shaded, but with the light of the sky coming through them. Such symptoms are found in trees affected by different strains or varieties of the virus that have several degrees of virulence.

Great caution is necessary to avoid mistaking injuries for psorosis symptoms. Thrips, aphids, mites, small hailstones, or minute sand grains driven by wind may all produce markings of similar size on young leaves.

Occasionally symptoms occur on mature leaves and fruit (fig. 2), especially in advanced stages of the disease. On water sprouts, leaves, and fruit, these appear as circular spots or rings that may become sunken and necrotic.

Psorosis is indicated by at least two types of bark symptoms (fig. 3): a localized type and a rampant type, which is rare. Similar young-leaf symptoms accompany both types and, especially with the rampant form, some leaves may show large blotches or rings that may become necrotic. Necrotic rings on fruit are occasionally found associated with psorosis.

The more common type usually begins either as scales of bark, with or without gum formation, or as aggregations of small pustules under which are brown specks. The scales of outer bark are dry, irregular flakes, 1/12 to 1/8 inch thick, with live, tan to buff-colored bark underneath. This form usually occurs first in localized areas on some of the older bark of trunks or limbs of trees six to 12 or more years of age. As the scaling advances, the deeper layers of bark and even the wood become visibly affected, even though still alive. Small or large amounts of gum often form, depending

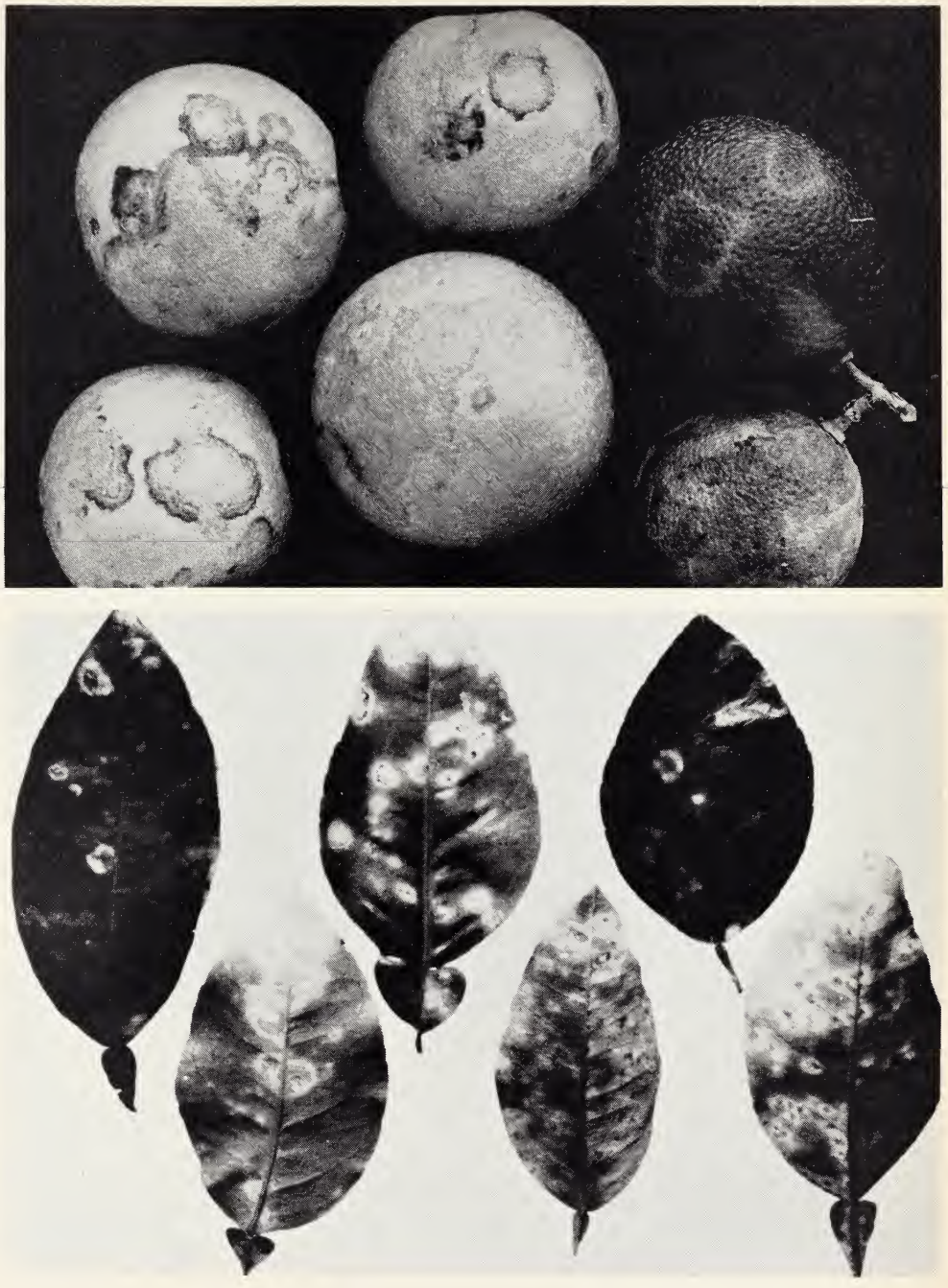


Fig. 2. Psorosis ring-type symptoms on navel orange fruit and on grapefruit leaves.

Fig. 3. Psorosis (scaly bark) localized form (left) and rampant form (right) on sweet orange.

on weather and growth conditions. The rate of scaling varies on different trees. After the lesion has been present for some years, gummy deposits occur beneath the bark and between the growth layers of wood. Later the wood dies and becomes light drab to brown or reddish-brown; the discoloration progresses in an irregular fashion, not necessarily following the grain of the wood. The affected portions of the tree then deteriorate rapidly and have only a few small, yellow leaves. The anatomy of psorosis lesions has been described (*20*).

The rampant type of common psorosis occurs infrequently, and differs from the usual, more localized type by showing gum formation in advance of, rather than after, scaling. It also may show a rapid advance of scaling in a continuous strip or area, usually along one side of the trunk or branch and even into much smaller limbs and twigs than is normal for the common form of psorosis. The

rampant type probably is not caused by a distinct strain of psorosis virus, for it can be produced by grafting a piece of bark from a localized psorosis bark lesion into a healthy orange tree (*73*).

No treatment other than pruning or tree removal is known for advanced psorosis. Treatment of beginning bark lesions of psorosis consists of scraping the bark, only before the tree shows any visible deterioration in the top, in order to prolong its usefulness (*28*). No permanent cure is known. After noticeable deterioration has set in, it is not as a rule profitable to treat the malady. When lesions are small, not larger than one's hand, scrape deep enough to eliminate discolored layers. Scrape surrounding bark, not visibly affected, for a distance of 6 to 8 inches above and below and 4 to 6 inches on each lateral margin. To disinfect the scraped area, use either potassium permanganate—about 1 per cent or 1 teaspoon of crystals to a pint of water—or other safe disinfectant. If scraping is done in dry weather it is not necessary to treat the scraped area, but the scraping tool should be disinfested before each tree is treated to avoid spreading other virus or fungus diseases. Declined branches may be pruned out. In scaling areas covering more than one-third the circumference of the trunk, it is not possible to scrape deeply enough to eliminate all discoloration. As soon as deterioration of the top begins, further treatment is usually uneconomical. A rapid chemical treatment for scaly bark developed in 1944–1946 was abandoned because its use necessitated extreme care to avoid injuring healthy bark and because production of the chemical "dinitro" (dinitro-o-cyclohexylphenol) was stopped (*36*).

Scraping or chemical treatment apparently delays the staining and plugging that cause rapid deterioration of the tree, but precise data for effects of treatment on production are not available.

The obvious step in controlling psororis and some other virus diseases of citrus is to use only virus-free plant materials for propagation. This does not assure the grower that trees will remain free of all virus diseases, since insect vectors can carry some viruses that may infect even carefully selected materials. The method is effective, however, against psorosis and other diseases that are transmitted mostly by union of living tissues, such as artificial budding and grafting, or natural root grafting, and against vectored diseases in areas where vectors are absent. Psorosis virus is rarely transmitted through seeds or by root grafting, and apparently has no vector in California. In Argentina, eruptive gummosis, a disease with symptoms similar to those of psorosis, appears to be spread by an unknown vector (*23*). The infrequency of psorosis virus transmission by natural root grafting is indicated by an experiment in which a row of psorosis-affected trees set out at Rancho Sespe, California, 40 years ago, at the normal planting distance of 22 feet from a row of healthy trees, has not yet transmitted the disease to the latter.

Many nurserymen, under the procedure specified by the California Department of Agriculture, produce and sell trees from budwood sources registered or certified as apparently free of psorosis and several other viruses. Registration is based on absence of the leaf symptoms described above and of the psorosis bark disorders described in the following pages, and indexing, on sensitive indicator plants, for psorosis (*24*) and other viruses. Planting only trees known to be free of any forms of psorosis virus is the way to avoid the losses caused by this important group of maladies.

BLIND POCKET

Blind pocket, until recently, was reported only in California (*35*) but is now known to be in Florida (*51*) and Brazil (*53, 57*), and is very common in Chile (*60*) and in some orchards of the

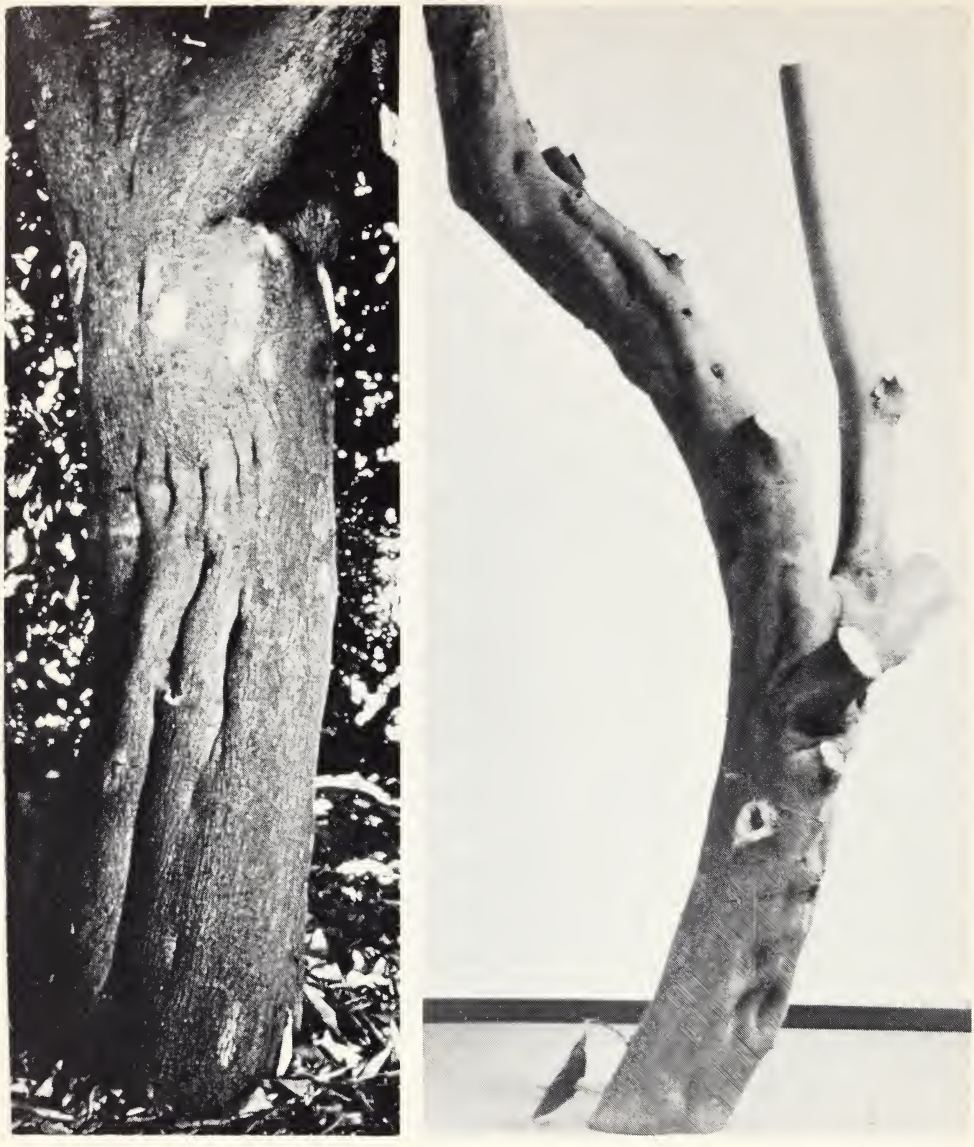


Fig. 4. Blind pocket disease of sweet orange.

Mediterranean basin (*6*). It is associated with young leaf symptoms similar to those seen with common psorosis. Blind pocket has two forms, one with plain blind pockets in bark and wood; in the other the concavities are accompanied by course, psorosislike eruptions. The eruptive form may result from infection by two or more viruses of the psorosis group. Blind pocket usually differs from concave gum disease (see page 11) in that it has narrower, more abruptly depressed, troughlike regions, frequently with two almost straight or even convex sides coming together at an acute angle at the bottom and top (fig. 4). Some cases show longer or shorter depressions with less abrupt slopes more nearly resembling the concavities of concave gum disease. Under each depression is an ocherous-salmon core of tissue that eventually is impregnated with a hard, gum-

like substance. Only rarely is gum forced out to the surface from these blind pockets. Within an area occupied by several blind pockets an eruptive lesion may occasionally occur, somewhat resembling a lesion of psorosis, but with thicker bark scales. No effective treatment is known for blind pocket.

CRINKLY LEAF AND INFECTIOUS VARIEGATION

It is generally agreed that crinkly leaf and infectious variegation are caused by strains of the citrus infectious variegation virus.

Crinkly leaf has been found especially in lemon trees. In addition to the flecking in young leaves characteristic of psorosis, blades of mature leaves show warping and pocketing on certain branches or over the entire tree (fig. 5). The fruit is frequently, but not always, coarse, rough, and misshapen on affected trees or branches. In severe cases, fruits are covered with irregular bumps. Somewhat similiar temporary effects may result from improper nutrition, scarring by the *Botrytis* fungus, or from mites, aphids, or other insects. Another type of roughened fruit appears to be an inherent bud variation. Workers in several countries have reported that purified preparations of citrus infectious variegation or crinkly leaf virus are highly infectious (*6, 20, 23*).

Infectious variegation has been found a few times associated with a crinkly leaf condition of lemons (fig. 5). Portions of the leaf blade of some leaves lack green, and are white to yellow. In some leaves the chlorotic areas are entirely on one side of the midrib and in others they are scattered over the entire blade but not arranged in any definite pattern. Some leaves near affected ones appear entirely normal. The young leaves also show flecking symptoms like those of psorosis.

The virus strains causing infectious variegation and crinkly leaf have been transmitted by budding to healthy lemon, sweet orange, and sour orange trees. Trees that have crinkly leaf or infectious variegation should not be topworked because the virus will enter the new scion and produce the same effect in the new top. Infectious variegation virus has been transmitted mechanically from citrus to other citrus and to herbaceous plants (*6, 35*). Sap inoculations with a mixture of infectious variegation and psorosis viruses transmitted only infectious variegation (*26*).

SATSUMA DWARF

This disease is of economic importance on citrus in Japan (*23, 53*). Many leaves of affected trees are abnormally small and narrow, and curled downward along lateral margins. Shortened internodes of twigs produce a dwarfed, bushy effect. Environment influences the type of leaf symptoms produced. Narrow, boat-shaped leaves are produced at cool to moderate temperatures (*24*), whereas small, dwarfed leaves (spoon-shaped) are produced at warmer temperatures. The small fruit is of poor quality with thickened rind at the stem end. These symptoms appear only on the spring flush of growth, not on the summer and fall flushes. The virus is readily transmitted to healthy citrus by budding and grafting. It is also mechanically transmissible from extracts of infected citrus and herbaceous hosts to healthy plants of host species. A number of legumes and white sesame are good indicator plants, showing, after a few days, chlorotic spots which become necrotic, with a yellowing halo. Infection then spreads into the mesophyll and kills the leaf.

While the tristeza virus (p. 13) is generally demonstrably present in Satsuma trees in Japan, Satsuma dwarf virus can usually be separated from it by passing inoculum from Satsuma dwarf-affected trees through trifoliate orange, which is resistant to the tristeza virus but suscepti-

Fig. 5. Crinkly leaf and infectious variegation: top, sour orange; bottom, lemon.

ble to the Satsuma dwarf virus. This indicates that Satsuma dwarf disease is caused either by a distinct virus or by that virus together with tristeza virus. Moreover, while Satsuma mandarin in Japan is generally a carrier of tristeza virus, the Satsuma dwarf disease is relatively uncommon in Japanese plantings.

Fig. 6. Concave gum disease symptoms: (left) on trunk and branches of sweet orange; (right) on orange leaves.

Possible relationships between Satsuma dwarf virus and citrus infectious variegation virus are suspected (*20*).

CONCAVE GUM DISEASE

Concave gum disease, reported in many countries, may show symptoms similar to those of common psorosis on young leaves—that is, small, clear areas or flecks in the region of the small veinlets. The relationship between concave gum virus and psorosis virus may not be as close as originally believed (*23, 53*). The oakleaf or watermark pattern is a distinguishing symptom (fig. 6). Most conspicuous are the broad concavities of various sizes on the trunk or limbs formed by the arrested or retarded growth of wood tissue in localized regions (*35, 62, 82*). These are for the most part covered with normal bark, with a crevice or crack through which gum may ooze to the surface at certain seasons. Under the bark of these concavities are masses of gummy, granulated tissue often cinnamon to tawny or

russet in color (*28*). The underlying layers of wood, which are usually thinner than normal, are impregnated with a semiliquid to cheesy or gummy substance. A few to many successive gum-filled layers of wood, alternating with layers of nearly normal wood, are found. The injurious effects may develop slowly over many years, but in time numerous lesions on a tree may result in serious deterioration. No effective treatment is known for concave gum disease.

DWEET MOTTLE

When a certain selection of Cleopatra mandarin (Citrus Research Center #270) was indexed on Dweet tangor it was noted that leaves of the latter developed a type of mottle that slightly resembled some patterns caused by psorosis or concave gum viruses (*23*). However, the mandarin, which showed none of these three types of symptoms, had twig dieback and produced little new growth and only small fruits. Further indexing with approved indicator plants showed that the mandarin was free of other known viruses except a mild strain of exocortis.

Other common commercial citrus varieties inoculated with Dweet mottle virus showed no symptoms but could act as carriers. When plants were inoculated with both the Dweet mottle virus and any other citrus virus, symptoms of the latter were not increased.

Moreover, since Dweet mottle virus did not protect sweet oranges against psorosis, concave gum disease, and infectious variegation-crinkly leaf viruses, and visibly affected only Dweet tangor, it is considered distinct from any previously reported. However, Dweet mottle virus has not yet been observed causing damage to commercial citrus.

Fig. 7A. Below: Tristeza (quick decline) of Valencia orange on sour orange.

TRISTEZA (QUICK DECLINE) of SWEET ORANGE on SOUR ORANGE and CERTAIN OTHER COMBINATIONS

The malady now known as tristeza, also called quick decline or bud union decline, or *podredumbre de las raicillas*, has been killing trees in California since as early as 1939 (*5*). By 1942 it was noted that the affected sweet orange trees were on sour orange rootstocks. In the early days of the industry in South Africa and Java, failure of trees on sour orange was thought by some to be a matter of horticultural incompatibility. Later, in South America, the fact that sweet orange on sour orange stock appeared to do well when grown in glasshouses, but soon failed when placed outdoors, led investigators to believe that the trouble was caused by a virus carried by insects. The nature of the disease was indicated when the virus was transmitted

in Brazil by means of the aphid *Toxoptera citricidus*, and in California by tissue grafts and by the cotton or melon aphid (*Aphis gossypii*) (27). In Florida, *A. spiraecola* and *T. aurantii* were also found capable of transmitting the virus (*48, 51*), and other vectors have been reported from India (*20*).

Flexuous, threadlike rods, about 10 to 12 nm × 2000 nm, are consistently present in the phloem of tristeza-infected citrus and are believed to be the cause of tristeza (*2, 23*). The existence of different strains of tristeza virus has been reported by several workers (*23, 53, 74*).

The disease has caused serious losses of susceptible citrus throughout much of the world with the exception of most countries in the Mediterranean basin, Mexico, and several countries of Central and South America. It is present worldwide, however, in most Meyer lemon trees and in certain tangerine selections. The infected trees in some countries and in some major areas of Arizona and of California, such as the San Joaquin and Coachella valleys, are being eradicated. Because of the high proportion of trees on sour orange rootstock in the Mediter-

Fig. 7B. Below: Tristeza symptoms on leaves (top) and wood (right) of Mexican lime indicator plant. Bottom, seedling yellows of Eureka lemon. Three seedlings at left are diseased; healthy control plant at right.

ranean areas, the disease could cause calamitous losses there. It has already caused losses in many orchards in Spain. Estimates indicate that tristeza destroyed about 25 million trees on sour orange in South America and about 3 million in southern California.

Symptoms of trees with tristeza (fig. 7A, B) are similar to those of trees in which roots have been injured or killed either by girdling due to gophers or by oak root fungus (*Armillaria*), or damaged directly by waterlogging, high concentrations of fertilizer, by kerosene or orchard heater oil, or by plant-growth regulators. The first noticeable symptom in the aboveground parts is a dull, ashen color and curling of the leaves lengthwise and upward (*3*). In the early stages, affected trees frequently have a large crop with many inside fruits. Diseased young trees usually begin to fruit one or more years before healthy trees of the same age. New growth in the fall is sparse, and navel orange fruits develop high color more rapidly than do fruits from healthy trees.

Below-ground symptoms precede those above ground. Even before anything abnormal is evident in the top, the small, fibrous roots at the periphery of the root system die, and their bark sloughs. Even in the early stages, the iodine test reveals a reduction in and, frequently, disappearance of starch in apparently otherwise unaffected roots. The process of starvation and destruction of roots proceeds from the tips inward toward the trunk of the tree, affecting more and larger roots. Microscopic examination (*20*, *63*) reveals that the primary diagnostic symptom of tristeza is the presence of darkly staining material in the clear cytoplasm of parenchyma cells, called chromatic cells, adjacent to the sieve tubes (food-conducting tubes). Next, these food-conducting tubes in sour orange bark near the bud union are destroyed, which accounts for symptoms resembling girdling. At some stage following destruction of sieve tubes, symptoms begin to appear in the top.

Generally the deterioration in the top is a gradual curling, wilting, and drooping of leaves and dying back of twigs, the process apparently keeping pace with root destruction. Frequently, however, during periods of low humidity, particularly those accompanied by a soil-water deficit, the trees (especially young ones) will collapse, and the leaves suddenly wilt and dry on the tree. This effect suggested the name "quick decline." Most trees so affected die. Others affected with the chronic form of the disease continue to produce. They retain too few functional sieve tubes for normal growth, however, but are able to live, commercially unproductive, for several years. The leaves and roots make feeble growth. Starch reappears in the roots if new, functional sieve tubes are formed. Another symptom frequently found is a netting, pinholing, or honeycombing of the inner bark of the sour orange rootstock of affected trees. The symptom is indicative but not diagnostic for tristeza.

A good indicator plant for diagnosis of tristeza is Mexican lime (*24*). When seedlings of this are budded with budwood from an infected tree, some of the newly forming lime leaves develop fleck-like vein clearings readily seen by transmitted light. The diseased leaves are also often cupped and chlorotic (fig. 7B). Wood of affected lime stems develops pits and grooves (*74*) (fig. 7B). Similar stem pitting is caused by tristeza virus in many citrus varieties, including Pera sweet orange (*53*).

In addition to quarantine, suppressive measures, and propagation only from tested, disease-free trees in clean or lightly affected areas, the only practical suggestion for control known at present is to avoid using sour orange and other susceptible rootstocks for sweet orange varieties, grapefruit, or tangerines. Susceptible rootstocks include *Citrus macrophylla*, sweet lime, citremon, grapefruit,

some tangelos and shaddocks, and, in some areas, citranges and trifoliate orange. *C. macrophylla*, now one of the most popular rootstocks for lemon in California, is highly susceptible to tristeza and, when exposed to aphids carrying tristeza virus, is likely to acquire the virus in the seedbed or nursery. Prevention consists of use of virus-free budwood, production of seedlings and nursery trees in tristeza-free areas, and prompt removal of *C. macrophylla* suckers from orchard trees. In some areas, where the virus is infrequently found in lemon trees, the infected lemon trees should be eliminated (*8*).

The rootstocks now in use for orange trees in areas where tristeza is present include sweet orange, Cleopatra mandarin, Rough lemon, Troyer and Carrizo citranges, trifoliate orange, and Rangpur lime (in South America). Of these, only the trifoliate oranges show much resistance to *Phytophthora* gummosis, foot rot, and root rot, which are of great importance especially where basin and flooding types of irrigation are practiced. Troyer citrange is intermediate in resistance to *Phytophthora* infection. The resistance of some trifoliates to both tristeza and *Phytophthora* offers a solution, provided scion budwood free of exocortis virus can be obtained. Some trifoliate selections, however, are known to be very susceptible to lime-induced chlorosis. Recently Troyer and some other citrange rootstocks have shown considerable susceptibility to tristeza in certain citrus areas of California (*12*). Use only true-to-type seedlings of trifoliate and citrange selections known to be tristeza-resistant in any tristeza-infested areas where the trees will be planted. The performance of 77 tristeza-resistant rootstocks in Brazil has been reported (*53*).

Heat treatment was successfully used to obtain tristeza virus-free budwood in Florida and California experiments (*74*). New branch growth free of tristeza and psorosis viruses was secured by exposing infected Mexican limes to 95° to 110°F in a heat chamber for 40, 78, or 107 days. Young tissue free from the tristeza-seedling yellows virus complex was obtained from lime seedlings grown for four weeks at 100° to 104°F (*74*). This virus complex has recently been eliminated from some plants of Meyer lemon and some other selections by heat treatment (*54*).

Seedling yellows is a virus disease found only in association with tristeza, and may be caused by one or more forms of the tristeza virus (*74*). It causes a stunting and chlorosis of seedlings of sour orange, grapefruit, Eureka lemon, and citron (*45, 72, 74*) (fig. 7B). Leaves of severely affected plants are small, greenish-yellow in color, and remain underdeveloped. Internodes of the stem and shoots are short, and multiple buds may appear at the nodes. The virus that produces these effects on the seedlings apparently occurs naturally in sweet orange and mandarin trees affected by tristeza in Australian orchards, and can be transmitted to seedlings by grafting. The orange and mandarin trees that were reservoirs for the virus suffered no serious injury if they were on their own roots, but were damaged if they were on sour orange or grapefruit rootstocks. Under natural conditions for infection by an insect vector, lemon as a scion escapes severe injury.

South African and American investigators offered the possible explanation that tristeza is caused by two disease-producing entities, one that causes the tristeza stem pitting and one that causes stunting and yellowing of the seedlings. In Australia the seedling yellows virus is known to be generally present in sweet orange and mandarin orchards of coastal New South Wales (*23, 74*) ; in California it has been found in Meyer and South African lemon, Satsuma and some other mandarins, Waialua orange, and kumquat (*72*) ; and it is widespread in Japan (*81*). Sweet orange trees naturally in-

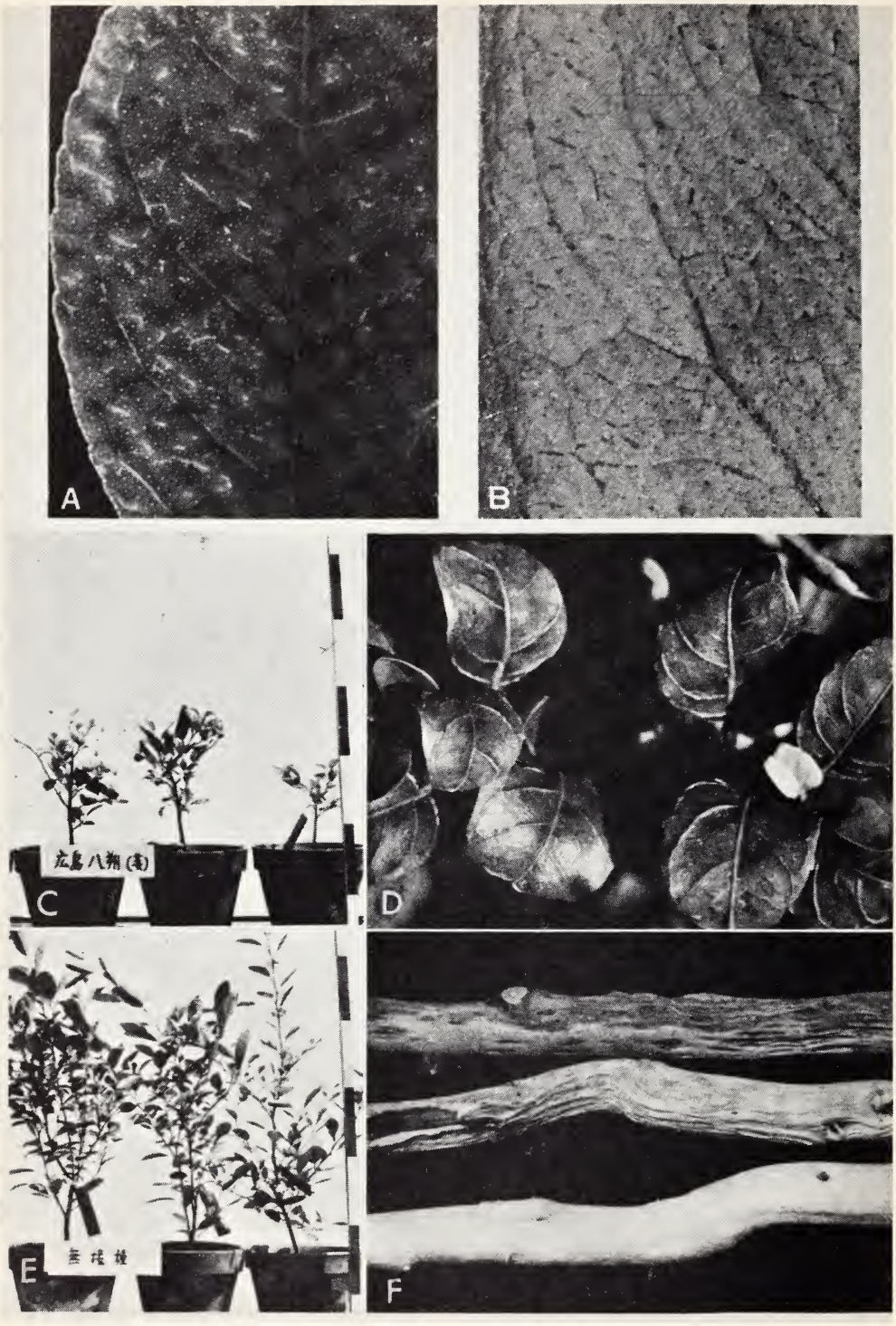


Fig. 8. Hassaku dwarf—effect on Mexican lime seedlings. A and B, vein clearing; C, stunted seedlings; D, vein corking and cupping of leaves; E, healthy seedlings; F, stem pitting. (Photos by Dr. Shoichi Tanaka.)

fected by seedling yellows virus have not been found in California.

While control measures suggested for tristeza should also apply here, it is recommended that propagation from seedling yellows trees be avoided in North American, Mediterranean, and other areas where the virus is not already well established in commercial plantings.

HASSAKU DWARF

This disease (fig. 8) of *Citrus hassaku* differs from Satsuma dwarf (p. 9) in that it does not produce symptoms on legumes or sesame, but causes vein corking and stem pitting and retards growth of Marumera (*C. obovoidea*) (*53*). Leaves of diseased trees are small, chlorotic, and wrinkled, and the leaf margins fold upward, producing a spoonlike appearance. Bark of branches and trunk becomes rough and bumpy and develops vertical concavities, with corresponding pitting in the wood. Mexican lime plants grafted with material from dwarfed Hassaku trees develop vein clearing, vein corking, and stem pitting (*61*), indicating that the disease may be caused by a strain or complex of tristeza virus probably similar to that involved in stem pitting of grapefruit (*30, 31, 38, 47, 53*). (See table 1.) *C. hassaku* on the resistant trifoliate rootstock is very susceptible, and on Rough lemon, severely stunted, possibly implying that a pathogen in addition to tristeza virus is involved. The Hassaku dwarf pathogen spreads in the orchard and apparently *Toxoptera citricidus* transmits it to various species of citrus (*20, 47*). It is obvious that propagation from Hassaku dwarf-affected trees should be avoided.

VEIN ENATION and WOODY GALL

Vein enation and woody gall are caused by a virus that has become widespread in citrus in southern California except in the eastern section (*75, 76*). The symptoms that give the disease its name are the small enations or lobes of tissue

Fig. 9. Woody gall, vein enation on Rough lemon. (Photographed from specimens supplied by J. M. Wallace.)

or galls that develop on the undersides of leaves, with corresponding depressions on the upper surfaces, and the gall-like growths that form on the roots, trunk, branches, and twigs. The vein enation symptom is rarely seen except in young plants, and woody gall is not common in most orchards, although under some conditions large woody galls develop near the bud union on rootstocks of Rough lemon and *Citrus volkameriana* (fig. 9). Such galls are common on Rough lemon rootstocks in Peru (*20*). Woody galls are characteristically lightly pitted on the cambial surface of the wood by numerous undeveloped or latent buds and, if aboveground, the cambial surface is light green. The galls tend to form near thorns and at graft unions or wounds on susceptible plants (*24*). The green peach aphid, *Myzus persicae*, and the cotton aphid, *Aphis gossypii*, are vectors (*24*,

Disease (common name)	Literature*	Symptoms	Indexing plants
PSOROSIS (Scaly bark)	*20, 23, 24, 36, 73, 74*	Clearing flecks along veinlets; local or general bark lesions, occasional ring type marks on mature leaves.	Sweet orange, mandarin, Mexican lime, *C. excelsa*, sour orange
BLIND POCKET	*35, 53*	Abrupt concavities on trunk; occasionally coarse shelling lesions.	Mandarin, sweet orange, lemon
CRINKLY LEAF	*20, 23*	Distorted crinkled leaves; flecking and stippling in young leaves.	Mandarin, sweet orange, sour orange, lemon
INFECTIOUS VARIEGATION	*26, 35*	See crinkly leaf; plus variegation in leaves.	Mandarin, sweet orange, sour orange, lemon
SATSUMA DWARF	*20, 23, 24, 53*	Leaves bent downward, boat-shaped or cupped (spoon-shaped); psorosislike leaf symptoms.	*Sesamum indicum*
CONCAVE GUM	*23, 53*	Oak-leaf pattern and flecks in young leaves, concavities on trunk.	Mandarin, sweet orange
DWEET MOTTLE	*23*	Psorosislike mottle on leaves.	Dweet tangor
TRISTEZA (Quick decline)	*2, 5, 8, 12, 23, 24, 27, 30, 31, 45, 48, 53, 54, 63, 72, 74, 81*	Veinlet clearing in young leaves; necrotic sieve tubes at bud union; honey-combing (inverse pits); decline of top. Pits in wood of lime. Chromatic cells in various hosts.	Mexican lime, *C. macrophylla*
SEEDLING YELLOWS	*23, 45, 72, 74, 81*	Stunting and yellowing of foliage of sour orange, grapefruit and lemon; stem pitting of Mexican lime.	Lemon, sour orange, grapefruit, Mexican lime
STEM PITTING	*23, 38, 47, 53*	Pits and grooves in wood of trunks and branches; decline of top with small mottled chlorotic leaves; tree stunted; small acid fruit, low production.	Grapefruit, Mexican lime
HASSAKU DWARF	*20, 47, 53, 61*	Small, pale, upfolded leaves; furrows or concavities on bark; stem pitting.	Marumera (*C. obovoidea*) and Mexican lime
VEIN ENATION, WOODY GALL	*20, 24, 42, 44, 75, 76*	Undersurface of veins with small projections (enations); corresponding concavities on upper surface of young plants; woody galls on trunk, branches, and roots.	Enations and galls on sour lime, *C. volkameriana*, some mandarin-limes, Rough lemon. Enations only on various other hosts.

*See "Literature Cited," p. 39.

Distribution	Vector	Other means of transmission	Varieties attacked	Control
Worldwide	None yet found	Budding, grafting, natural root grafting, sap, dodder	Leaf and bark symptoms; sweet orange, grapefruit, mandarin, tangelo; only leaf symptoms on most others	Use disease-free budwood and rootstock. Scrape lesions and remove bad branches.
Worldwide	None yet found	Budding, grafting, natural root grafting	Sweet orange, lemon, mandarin	Use disease-free budwood and rootstocks.
Worldwide	None yet found	Seed (rarely), budding, grafting, natural root grafting, sap	Sweet orange, lemon, sour orange	Use disease-free budwood and rootstocks.
Worldwide	None yet found	Budding, grafting, sap	Lemon, sour orange	Use disease-free budwood and rootstocks.
Japan	None yet found	Budding, grafting, sap	Satsuma mandarin	Use disease-free budwood and rootstocks.
Worldwide	None yet found	Budding, grafting, natural root grafting	Sweet orange, mandarin, lemon, lime, sour orange	Use disease-free budwood and rootstocks.
California	None known	Budding and grafting	Dweet tangor	Use disease-free budwood and rootstocks.
Nearly worldwide	*Toxoptera citricidus*, *T. aurantii*, *Aphis gossypii*, *A. spiraecola*, *A. cracovora* *Dactynotus jacae*	Dodder, budding, grafting	Sweet orange, mandarin, etc., on sour orange, grapefruit, and some other rootstocks; lime	Quarantine and eradication for very limited infestations. Control vectors. Use disease-free budwood and tolerant rootstocks, such as trifoliate, Troyer citrange, sweet orange, Rough lemon, Rangpur lime, Cleopatra mandarin. Lemon trees on various stocks tolerant to tristeza may be damaged by seedling yellows.
Nearly worldwide	*T. citricidus* *A. gossypii*	Dodder, budding, grafting	Grapefruit, lemon, sour orange, citron	Same as above.
Known in Aden, Uruguay, Brazil, Argentina, Southern Africa, Australia, Japan	*T. citricidus*	Dodder, budding, grafting	Grapefruit, *C. hassaku*, certain other varieties	Select for tolerance to stem pitting.
Japan	*T. citricidus*	Budding, grafting	*C. hassaku*, *C. obovoidea*, King mandarin	Use disease-free budwood. (Control vectors?)
U.S.A., S. Africa, Australia, Peru, Japan	*T. citricidus* *A. gossypii* *Myzus persicae*	Budding, grafting, dodder	Sweet and sour orange, Mexican lime, lemon, Rough lemon, *C. volkameriana*, etc.	Avoid Rough lemon, *C. volkameriana*, and sour lime rootstocks. Use disease-free budwood in clean areas.

Continued

Disease (common name)	Literature*	Symptoms	Indexing plants
EXOCORTIS	*1, 4, 23, 24, 33, 53, 54, 56, 68, 69, 74, 80*	Cracking and scaling of bark of trifoliates, citranges, and Rangpur lime; leaf and stem epinasty, cracking and callousing of underside of midveins, yellow blotches and cracks on shoots of some citrons; stunting of tree.	Etrog citron (USDCS No. 60-13; Arizona 861)
CACHEXIA-XYLOPOROSIS	*15, 23, 24, 53, 55*	Lens-shaped pits on trunk with or without gumming; pegs on inner bark surface.	Orlando and Wekiwa tangelos, Parsons Special mandarin, Palestine sweet and Kusaie limes.
CRISTACORTIS	*23, 54*	Pits in wood of trunk and branches; crested, gummy pegs on inner bark surface.	Orlando tangelo, sour orange
IMPIETRATURA	*20, 23, 24, 52*	Hard, gummy deposits in rind, central core, and vascular bundles of fruit.	Grapefruit
YELLOW VEIN	*16, 78*	Bright yellow veins of leaves.	Mexican lime, Rough lemon, Etrog citron USDCS 60-13
LEAF CURL	*53*	Dieback of branches, curling of leaves like aphid injury; excessive flowering, only few small fruits; gum and grooves in wood.	Caipira sweet orange
TATTER LEAF	*11, 17, 23, 24, 67, 77*	Blotching and distortion of young leaves and twigs.	*C. excelsa*
CITRANGE STUNT	*11, 17, 23, 24, 67, 77*	Stunting; deep pits and grooves in wood; blotched, distorted leaves.	Troyer, Carrizo, or Rusk citrange
RINGSPOT	*23*	Yellowish rings with green islands on some leaves, sometimes vein clearing and stem lesions.	Grapefruit, lemon, Rough lemon, sweet orange
STUBBORN	*9, 10, 14, 20, 21, 22, 23, 24, 25, 29, 34, 35, 49, 50, 52, 53, 54, 74*	Trees stunted; low yield. Distorted, eccentric, or "acorn"-shaped fruits, sometimes stylar-end greening and blue albedo; seed abortion; stiff, upright, multiple twigs and buds; small chlorotic or mottled leaves; premature defoliation; dieback in severely affected trees.	Sweet orange, tangelo
GREENING	*18, 19, 20, 23, 32, 41, 43, 46, 52, 53, 66, 71*	Similar to stubborn but usually with more chlorotic foliage and greening of fruit, dieback of branches; sometimes death of tree.	Sweet orange, tangelo
YELLOW SHOOT	*43, 52, 54*	General decline, yellowed or mottled leaves, premature defoliation, root decay, wilting, death of tree.	See Greening and Stubborn

*See "Literature Cited," p. 39

1 (*Cont.*)

Distribution	Vector	Other means of transmission	Varieties attacked	Control
Worldwide	Mechanically by cutting tools, fingers, and possibly by scratching and gnawing of animals	Budding, grafting, dodder, sap	Trifoliates, citranges, Rangpur lime, other mandarin limes, sweet limes, some lemons, citrons	Use seed and budwood from disease-free sources; avoid susceptible rootstocks. Sterilize cutting tools to prevent spread to clean trees.
Most citrus regions	None known	Budding, grafting	Sweet limes, mandarins, mandarin limes, tangelo	Use seed and budwood from disease-free sources; avoid susceptible rootstocks.
Mediterranean countries	None known	Budding, grafting	Grapefruit, sweet and sour oranges, mandarin, tangelo	Use disease-free budwood.
Mediterranean countries	None known	Budding, grafting	Grapefruit, oranges, mandarins, tangelos, lemon	Use disease-free budwood.
California (very limited)	None known	Budding, grafting	Limequat, kumquat, Mexican lime, calamondin, Orlando tangelo, Troyer citrange, lemon, citron	Use disease-free budwood. Destroy infected trees.
Brazil	None known	Budding, grafting	Sweet orange, lemon, grapefruit, citron, shaddock, tangerine, sweet lime	Use disease-free budwood. Destroy infected trees.
U.S.A. and probably in Meyer lemon in other countries	None known	Budding, grafting	*C. excelsa;* Meyer lemons tested found to be symptomless carriers	Use disease-free budwood.
In Meyer lemon in U.S.A. and probably in other countries	None known	Budding, grafting, sap	Citranges, citremon, other trifoliate hybrids, trifoliates	Use disease-free budwood. Destroy infected trees.
U.S.A., Central America, Mediterranean area	None known	Budding, grafting, dodder	Sweet orange, mandarin, lemon, grapefruit	Use disease-free budwood. Destroy infected trees.
Probably worldwide	None yet found	Budding, grafting	Sweet orange, sour orange, grapefruit, mandarin, tangelo, lemon, lime, Rough lemon, citron, most other varieties regardless of rootstock	Use disease-free budwood.
South Africa, India, Indonesia, Philippines, Taiwan, and probably most of S.E. Asia	*Trioza erytreae* *Diaphorina citri* (citrus psyllids)	Budding, grafting	Same as for stubborn	Use disease-free budwood. (Control vectors?)
China, Taiwan	*Diaphorina citri* (?)	Budding, grafting	Tankan and Ponkan mandarins, sweet orange, *C. grandis*, calamondin, and various others	Use disease-free budwood and rootstocks.

Fig. 10. Exocortis: (above, left) on trifoliate rootstock; (above, right) yellow blotches on trifoliate branch; (below, left) epinasty effect on Etrog citron, an indicator plant.

42). These insects transmitted vein enation virus to Mexican lime seedlings from which, by graft-inoculation, the virus causing woody gall in Rough lemon seedlings was transmitted, thus indicating the association of the two manifestations of this virus disease. Vein enation has been reported in South Africa, where it was shown to be transmitted by the aphid *Toxoptera citricidus* (*44*). Woody gall is present in New South Wales, Australia, and is bud-graft transmissible. Since nonbudded Rough lemon seedlings are affected in that area, an insect vector is assumed to be present and active. The economic importance of the disease has not yet been determined.

EXOCORTIS

Exocortis of trifoliate orange, citranges,

Rangpur lime, sweet lime, and citron is caused by a virus and is characterized by vertical splits in the bark and by narrow, vertical, thin strips of partially loosened outer bark (*4*, *23*, *53*, *74*). Yellow blotches often occur on young infected stems of these varieties (fig. 10). Troyer citrange is usually less severely affected than trifoliate orange. On some varieties of citron, exocortis causes epinasty, and cracking and darkening of veins and petioles of leaves. Orange, lemon, grapefruit, and other citrus trees on exocortis-sensitive rootstocks are slightly to greatly retarded in growth. Infected Eureka lemon on tolerant rootstocks is significantly retarded (*74*). The average yields of severely affected orange trees may be reduced as much as 40 per cent (*23*). Exocortis virus is present in most citrus areas and has increased in importance with the expanded use of exocortis-sensitive rootstocks. Exocortis virus is readily transmitted to healthy plants by budding knives (*33*) or other mechanical means and by hand (*56*). It is also transmissible from citrus to citrus by dodder (*80*). The virus is very stable, resistant to heat, and can persist on contaminated surfaces for many days (*1*,*69*). Exocortis virus separated from many of the host-cell contents can be inactivated by ribonuclease (*68*). This finding, together with the absence of typical virus particles in infectious preparations, led to the conclusion that this virus exists as a free ribonucleic acid (RNA) species in the host. There is some evidence for the existence of two infectious entities (*69*). Several factors were found important in the spread of the virus, including contaminated hands and tools of nurserymen and orchardists, which came into contact with phloem tissue in the bark; the fact that most citrus and many herbaceous plants can serve as donors of the virus; and the greater ease of transmissions between related plants than between unrelated ones (*54*).

For the detection of exocortis virus, a rapid indexing method is available which utilizes the virus-stimulated epinasty and stunting of Etrog citron selections, U. S. Date and Citrus Station # 60-13 or Arizona 861 (*24*) (fig. 10). Fluorescent chemical markers are useful in detecting exocortis virus in sensitive varieties (*23*). *Gynura aurantiaca*, *C. sarmentosa*, *Petunia* spp., and several other herbaceous plants exhibit pronounced epinasty, systemic mosaic, stunting, and necrosis when inoculated with exocortis virus (*80*).

To control exocortis use virus-free, registered budwood and sanitary budding, nursery, and field practices. Disinfest tools between cuts into different plants by dipping in a 10 to 20 per cent solution of household bleach (5.25 per cent sodium hypochlorite in the concentrated bleach). To retard corrosion of the cutting edges, rinse in a solution-emulsion of 1 part vinegar, 2 parts water, and 2 teaspoons of an emulsifiable spray oil in each pint (*56*).

CACHEXIA

Cachexia (xyloporosis) is a bud-transmissible virus disease which attacks the stem bark and wood of several citrus species and varieties (*24*, *31*, *51*). In this country the virus is frequently present in grapefruit and sweet orange without producing apparent symptoms, but it causes severe symptoms on *Citrus macrophylla* and many varieties of mandarin, tangelo, sweet lime and their hybrids (*6*, *15*, *23*, *24*, *53*). There is no evidence that the disease is carried through seed, and insect transmission has not yet been demonstrated.

The wood shows elongated pits and the bark produces growth ridges that fit into the wood pits (fig. 11). Brown, gummy deposits form in the pits and bark phloem, and the stem wood may eventually become brown. Bark scaling—which may not result only from cachexia—secondary organisms, and various nutritional deficiencies complete the

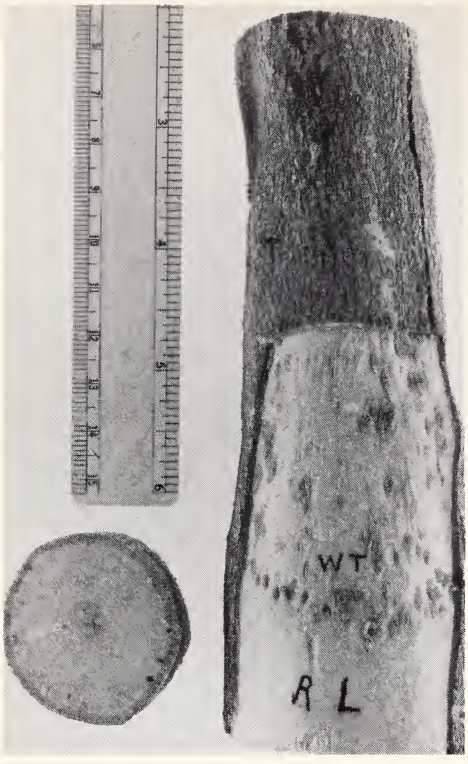


Fig. 11. Xyloporosis-cachexia: affected Wekiwa tangelo on Rough lemon rootstock.

degeneration of the tree. While the disease is uncommon in trees of young budlines, it is very important in Israel in trees of old budlines on Palestine sweet lime rootstocks, in which it produces a weakened, elastic wood (*55*), and it has recently appeared in some *C. macrophylla* rooted lemon trees in California.

Xyloporosis-affected trees in Israel are treated by inarching with sour orange. Many investigators are of the opinion that cachexia and xyloporosis are caused either by the same virus or strains of the same virus.

Control is a matter of prevention; that is, the use of tolerant scions and rootstocks or of budwood from indexed parent trees free of the virus. Indexing is done on susceptible indicator varieties, such as Orlando tangelo and Parsons Special mandarin, a method that often requires waiting two or more years for the development of typical symptoms (*24*).

CRISTACORTIS

Cristacortis is a severe, graft-transmissible disease causing stem pitting on sour orange (fig. 12), Tarocco orange, Orlando tangelo, and some other varieties in several Mediterranean countries (*23*). Cristacortis resembles, but is apparently different from cachexia, and causes a very severe reaction in Orlando tangelo, which is a good indicator plant (*54*). Since neither vector nor seed transmission is known, prevention depends on the use of clean, indexed propagative materials.

IMPIETRATURA

Impietratura, formerly believed to be

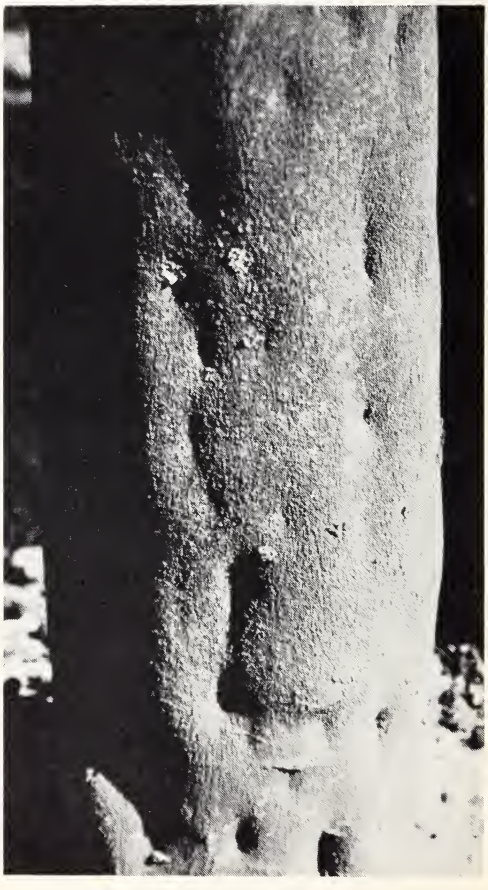


Fig. 12. Cristacortis pitting in sour orange.

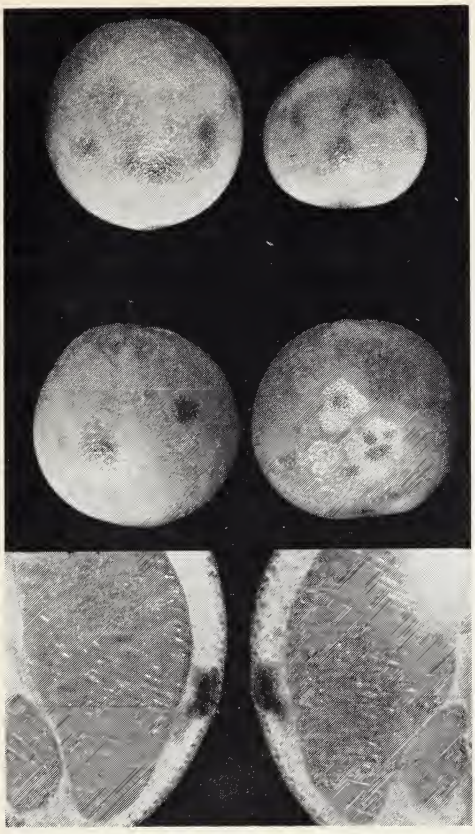


Fig. 13. Impietratura: gumming in navel orange peel. (Photographs by Henri Chapot.)

caused by a water deficit, is now known to be graft-transmissible and is presumed to be caused by a virus (*24, 52*). All of the commercial species of citrus are susceptible, and the disease is widespread in the Mediterranean region (*20, 23, 52*). The malady is characterized by small, more or less pear-shaped, hard fruits. The fruit has a hardened flavedo, and the rind generally has a few to many hard, gum-infiltrated protuberances (fig. 13) each surrounded by a yellowish halo. Many of the fruits on diseased trees fall off during the summer, the extent of loss being in direct proportion to severity of infection. The symptoms suggested boron deficiency as the cause, but analyses revealed adequate amounts of that element in the affected trees, and applications of boron compounds failed to correct or prevent the disease (*20, 53*).

The growth from healthy orange and grapefruit buds that had been placed in impietratura-affected trees soon developed typical symptoms of impietratura, and shield buds from diseased trees transmitted the pathogen to young, healthy grapefruit trees (*20, 24*). Transmissibility and incubation periods were highly variable. Sour orange and Avana mandarin are tolerant.

Use of tolerant varieties and of propagative material from disease-free trees is suggested as a control measure.

YELLOW VEIN

Yellow vein is a virus disease found on limequat, and is graft-transmissible to several rutaceous species, varieties, and hybrids, including West Indian lime, calamondin, Orlando tangelo, Troyer citrange, lemon, and kumquat (*78*). The striking symptom of the disease is the yellowing of the veins. The bright yellow color extends into the adjacent tissue and causes the veins to appear enlarged (fig. 14). The yellowing may extend down from a leaf petiole onto the young twig or stem. Certain citron, Red Rough lemon, and India lime selections are good indicators of yellow-vein virus (*16, 78*). Surveys thus far have indicated that the disease is not widespread, for it has been found in only four of many limequat trees in three California counties, and in one *C. macrophylla* seedling at Indio, California.

LEAF CURL

Leaf curl, reported only from Brazil, was shown to be transmissible by budding and grafting to several sweet orange varieties, Eureka lemon, sour orange, citron, grapefruit, and shaddock (*53*). It appears to be caused by a virus entity distinct and different from the exocortis virus. The main symptoms are a curling and distortion of the leaves resembling that caused by a heavy aphid infestation;

Fig. 14. Yellow vein of Mexican lime.

abundant production of weak sprouts with gum in their wood vessels near their unions with branches; and channeling and pitting of the wood of the trunk and main branches. Indexing can be done in two to five months on sweet orange seedlings. The obvious control in Brazil was to eliminate the few trees found infected.

TATTER LEAF and CITRANGE STUNT

Mexican lime and *Citrus excelsa* seedlings inoculated from Meyer lemon develop chlorotic blotches on their leaves, whereas the leaves of inoculated citrange and citremon plants become dwarfed, distorted, and strongly blotched, and the plants are stunted (figs. 15, 16). The effects are obtained whether or not the

tristeza-seedling yellows virus complex is present in the Meyer lemon, and indicate the presence of another virus (or viruses) at first named tatter leaf virus (*77*).

Additional studies revealed that the virus causing tatter leaf on *Citrus excelsa* is not the same as that which causes stunting of citrange (*23*). The latter virus was, therefore, designated as citrange stunt virus and is believed to be the virus transmitted mechanically from Meyer lemon to various hosts and to citrus from herbaceous hosts (*17, 23, 67*). The citrange stunt virus was purified and characterized (*67*). The tatter leaf virus is often lost during passage through *C. excelsa* or some other citrus hosts, and has not been transmitted mechanically,

Fig. 15. Left: Tatter leaf of Citrus excelsa. (Photo by T. A. DeWolfe; leaves from J. M. Wallace.)

Fig. 16. Below: Citrange stunt of Troyer citrange. (Specimen supplied by J. M. Wallace.)

whereas the citrange stunt virus appears to be retained indefinitely by many citrus and herbaceous hosts and is mechanically transmissible.

Citrange stunt virus causes such severe stunting and distortion of citrange seedlings that it could have considerable economic importance if it becomes widespread. It is assumed to cause a severe bud-union creasing, grooving, fluting, and stunting disease of Troyer citrange rootstock in trees inoculated with Meyer lemon buds (*11, 23*). No vector is known for either the tatter leaf or the citrange stunt viruses. Although neither virus has been found in any uninoculated trees other than Meyer lemon, the citrange stunt virus occurs in some specialty trees that have Meyer lemon as one component.

Indexing for tatter-leaf virus is done on *Citrus excelsa* seedlings. Citrange stunt virus may be indexed on Rusk, Troyer, or Carrizo citrange seedlings, but not on *C. excelsa* (*17, 24*). Cowpea is also an indicator plant for citrange stunt virus (*24, 67*).

CITRUS RINGSPOT

Leaves of sweet orange seedlings inoculated with tissue from a lemon tree with psorosis developed not only typical psorosis flecking but also yellowish spotting of leaves that later changed to ring patterns and coalescing blotches as the center area of the spots remained green (*23*). Vein clearing, resembling that of tristeza at first, spread into adjacent areas of tissue, resulting in a feathery effect (fig. 17). The leaf patterns, together with a shock effect of the disease, which caused leaf drop, stem lesions, and death of parts, suggested certain effects of the rampant form of psorosis. However, sweet orange seedlings infected with ringspot virus were not protected from psorosis infection, nor did infection with concave gum virus protect the seedlings against ring-

Fig. 17. Ringspot of lemon. (Leaves supplied by J. M. Wallace.)

spot. Previous infection with psorosis or blind pocket viruses did, however, protect against ringspot.

While to date ringspot has only occasionally been observed on orchard trees in California and may be of little or no economic importance, the leaf symptoms have been seen on field trees in Florida and Central America. With present knowledge, use of disease-free propagative material is the only recommendation for control.

MYCOPLASMALIKE DISEASES

STUBBORN DISEASE

Stubborn disease materially reduces fruit bearing in commercial citrus varieties and especially in navel oranges (*20*, *29*). It is believed to be caused by mycoplasmalike bodies and not by a virus (*34*). The little leaf disease of Israel appears to be the same as stubborn (*20*). The so-called "crazy top" of grapefruit trees with "acorn" or "pink nose" fruit, found in Arizona and California, is believed to be the same disease (*3*).

Stubborn disease often retards growth considerably in trees infected when young, results in excessive abortion of seeds in many seedy varieties, and usually

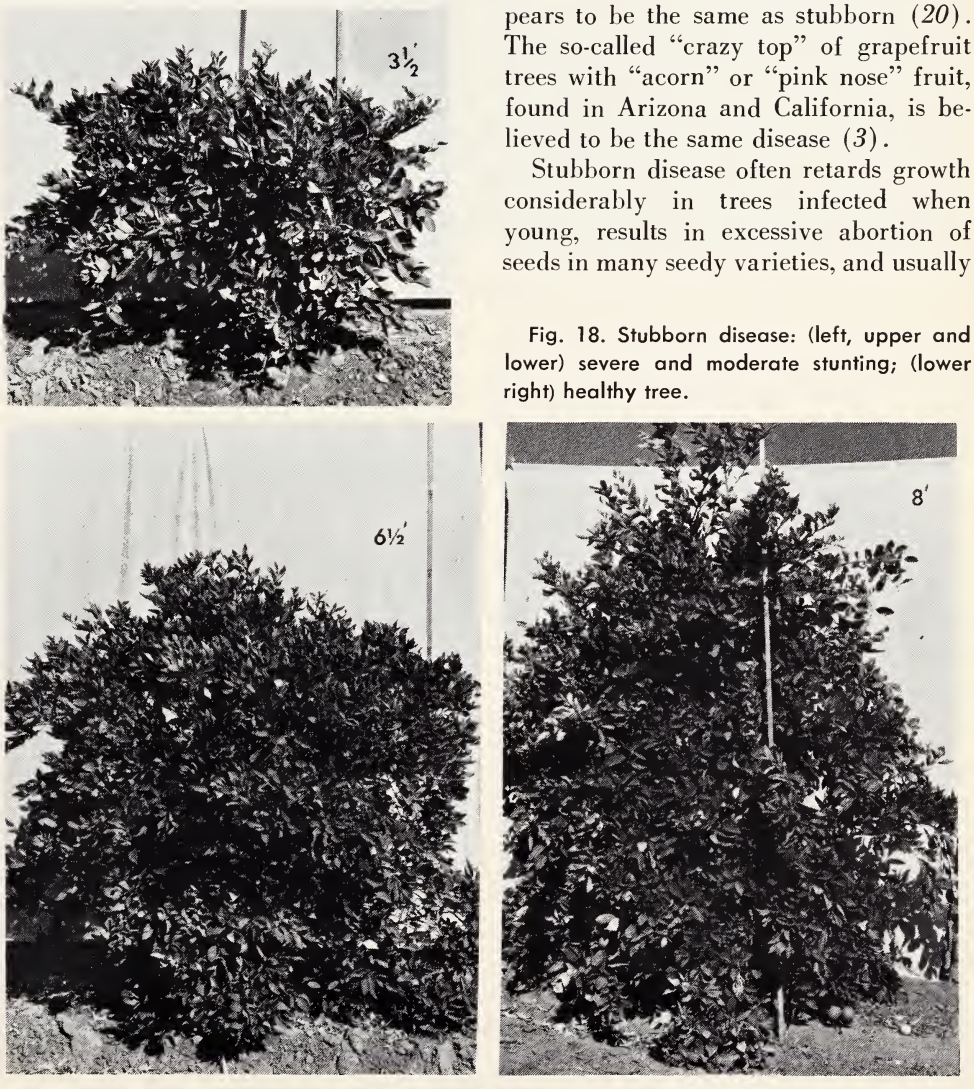


Fig. 18. Stubborn disease: (left, upper and lower) severe and moderate stunting; (lower right) healthy tree.

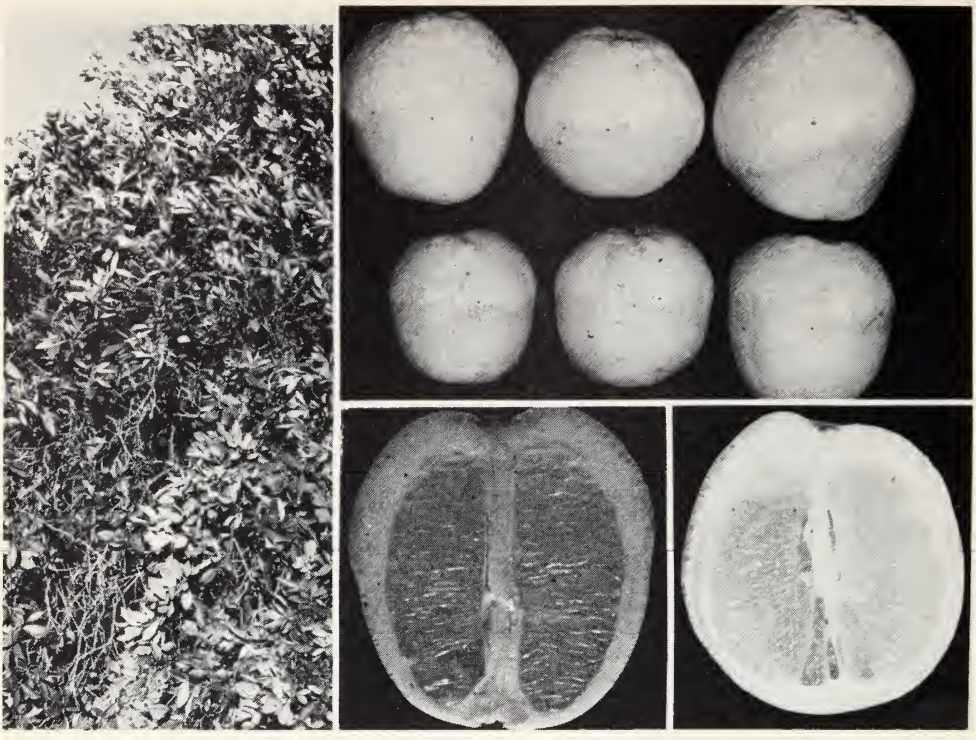


Fig. 19. Stubborn disease: affected foliage and fruit of navel orange.

causes bunchy, upright growth with leaves smaller than normal (*14*, *20*) (figs. 18 to 22). On navel orange the symptoms, when pronounced, include a brushlike growth of twigs as a result of abnormal branching from multiple buds. The leaves tend to bend upward more on each side of the midrib than do those of healthy trees. Growth flushes are frequently out of phase with those on healthy trees, and the greatest amount of shoot growth on the diseased trees often occurs in the autumn. Foliage of some or all shoots tends to be intermittently abnormally mottled and chlorotic. When fruits are produced, many are smaller than normal and may be lopsided and occasionally acorn-shaped. The rind in affected portions, usually on the sides and distal half of the fruit, is thinner than the rind of healthy fruits (figs. 19 and 21). In severely affected fruits the pulp below the thin portions of

Fig. 20. Stubborn disease: test with Madam Vinous sweet orange indexing plant, showing healthy seedling (largest), one mildly affected, one severely affected. Leaf symptom at right.

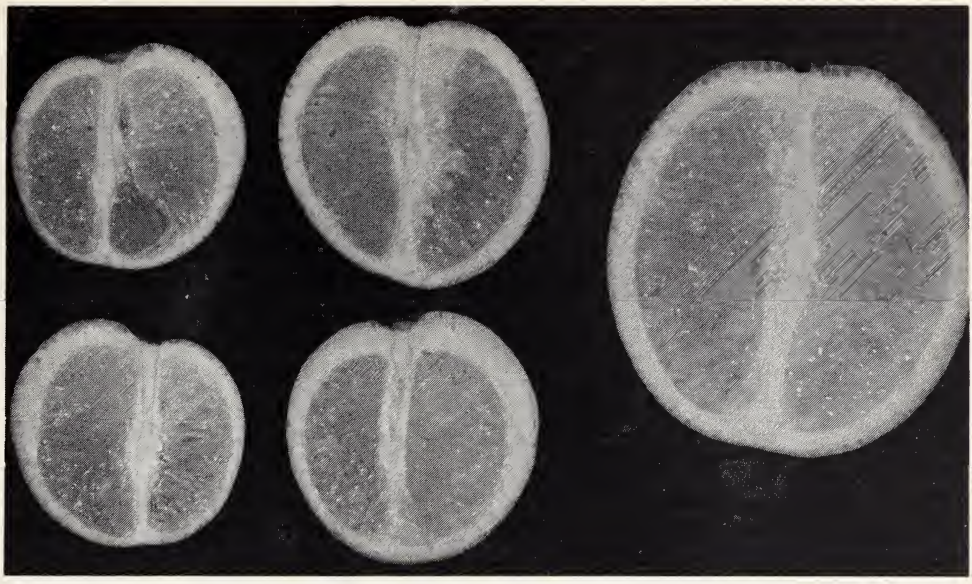
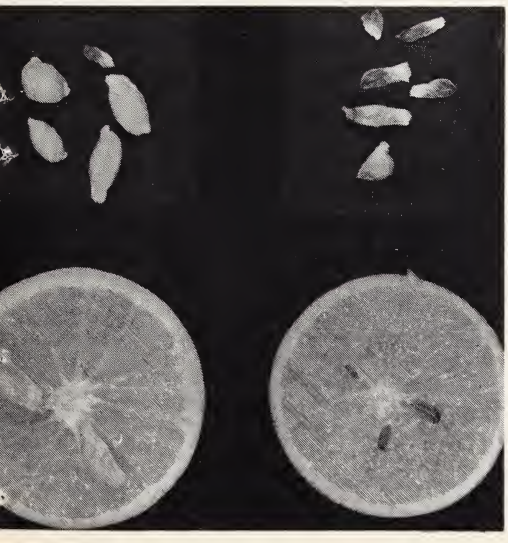


Fig. 21. Stubborn disease: Valencia orange fruit showing lopsidedness (four fruits on left) and healthy fruit (right).

the rind has a sour or bitter taste and disagreeable odor. The distal end of maturing fruits sometimes changes color from green to orange more slowly than does the button end, a reversal of the coloring process in normal fruits. The symptoms on the mature fruit are useful in diagnosis. The fruit symptoms of stubborn have been reproduced by graft inoculation of healthy trees and by grafting healthy fruits onto seedlings infected with the disease (*35*, *49*). Occasionally the albedo of diseased fruit has a blue color. Several detailed descriptions of stubborn disease and its effects on certain varieties have been published (*9*, *10*, *20*, *21*, *22*, *25*, *74*).

Fig. 22. Stubborn disease: (left) healthy Valencia orange and seed; (right) diseased orange and aborted seed.



In many species and varieties, especially navel orange, symptoms are highly variable, and several forms of the disease, possibly caused by different forms of the pathogen, are known. The stubborn pathogen is irregularly distributed in the tree, and certain branches of some stubborn trees are symptomless (*20*, *23*, *49*, *53*). While symptoms on large trees develop slowly, small trees and indicator plants develop symptoms rapidly but may improve later (*20*, *53*). The Madam Vinous variety of sweet orange and several others, Sexton tangelo, and some grapefruit varieties, grown at high temperatures, are suitable indicator plants for stubborn disease (*24*, *50*).

Repeated experiments corroborate many previous observations by propa-

gators and growers that trees with stubborn symptoms, when topworked with carefully selected, healthy buds or scions, again produce the same symptoms after developing a new top.

Stubborn disease is currently the greatest disease threat to production of sweet oranges and grapefruit in California. Although no vector has been found, the disease has been spread into many trees, apparently by natural means, and has been so widely disseminated by propagation that it is now one of the most important citrus diseases in California, Arizona, Morocco, Israel, and some other Mediterranean countries, is present in Mexico and several South American countries, and may be worldwide. For prevention of the disease in future plantings, it is most important that nursery trees be propagated from trees free of the stubborn disease pathogen. Examine parent trees very carefully, and cut budwood only from healthy trees and from branches that bear only normal fruits. Remove young stunted and stubborn-diseased trees and trees rendered unproductive by stubborn.

Fig. 23. Greening disease: (top) in Taikat mandarin on Calamandarin rootstock (Philippines); (bottom) leaf symptoms of mandarin (Hong Kong).

Fig. 24. Likubin or yellow shoot of mandarin near Hong Kong (left) and in Taiwan (right).

GREENING DISEASE

Greening disease is of major importance in South Africa, India, the Philippines, Indonesia, Thailand, Taiwan, and China. It is similar to stubborn disease in many ways, but has little or no tendency to cause brushlike growth or acorn-shaped fruit (*20*, *23*, *32*, *41*, *53*, *66*). Either affected branches or the entire tree is seasonally chlorotic with mottled leaves (fig. 23), and fruits are commonly of poor quality, retaining areas of green peel until maturity. Greening has been transmitted by the psyllid, *Trioza erytreae*, in South Africa (*46*), and in India and some other areas by another psyllid, *Diaphorina citri* (*13*, *18*, *19*). Greening has been the major factor in the devastation of orange orchards in some districts of South Africa, India, the Philippines (fig. 23), and Indonesia (*20*, *23*, *71*). Mycoplasmalike bodies have been found associated with greening disease (*41*).

"Yellow shoot" or "likubin," reported from China (*43*) and Taiwan (*52*), is evidently a form of greening or stubborn, despite its frequent association with tristeza virus (fig. 24). A fluorescent chemical marker, often present in infected trees, has been useful for detection of greening disease (*20*, *23*).

DISEASES OF UNKNOWN CAUSE WITH VIRUSLIKE SYMPTOMS

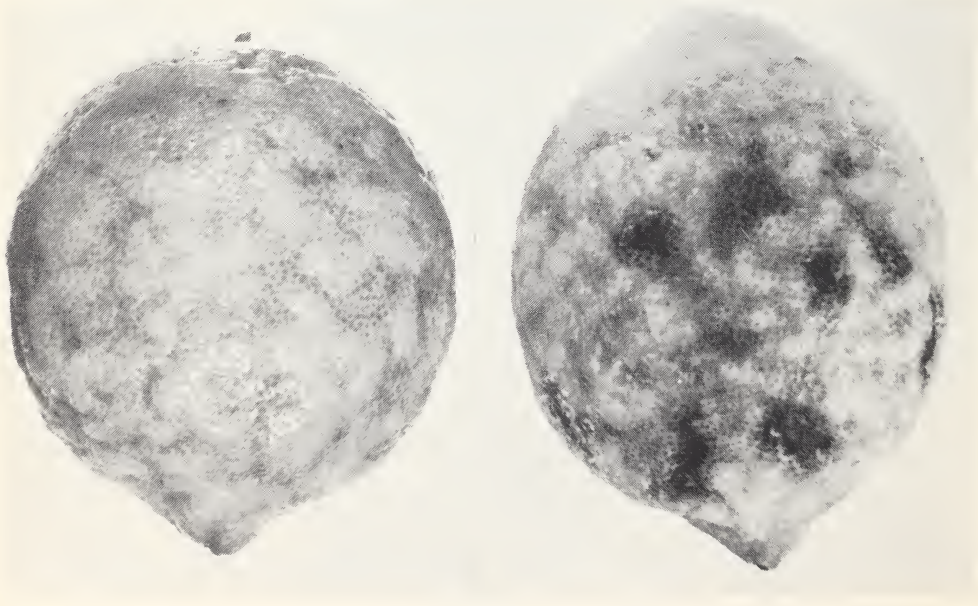
RUMPLE

A serious abnormality of lemon fruits in Florida was first reported in 1958 under the name rumple (*20, 40*). A disorder of Sicilian lemons, called wrinkle rind, and resembling rumple, was described in 1963 (*59*). It is now evident that the troubles in Sicily and Florida are similar. The disease is also known to be present in Turkey, Cyprus, Lebanon, and Ethiopia.

In Florida, rumple appears first in late summer as faint, chlorotic speckles on the rind surface, each spot covering an area including four or five oil glands. The oil glands first turn "greenish brown, then in order, tan, mahogany brown, and brownish black"; finally they collapse (*20*) (fig. 25). In Sicily the breakdown appears to be initiated always near the end of the summer or beginning of autumn. Green lemons show the effects during September, October, and November. After mid-December, or when a fruit starts its yellow coloring, it will not develop the trouble. When affected green lemons are placed in storage, the involved rind areas enlarge and new ones form, but losses from subsequent decay are not greater in rumpled than in normal fruits. While the disease ruins lemons for the fresh-fruit market, they may be used for juice, rind oil, and other by-products (*40*).

The importance of rumple has been indicated by extensive surveys. Thirty-eight of 40 lemon varieties examined in Florida showed an average incidence of the disease ranging from 0.7 per cent in the Harvey selection to 37.8 per cent in the Edwards selection (*20*). In Florida, the two varieties commonly grown in California—Lisbon and Eureka—had 8.8 and 8.4 per cent rumple, respectively. However, incidences of the malady in 1963 in some Florida orchards reached

Fig. 25. Rumple: (left) primary stage; (right) secondary stage. (Photos by Dr. L. C. Knorr.)



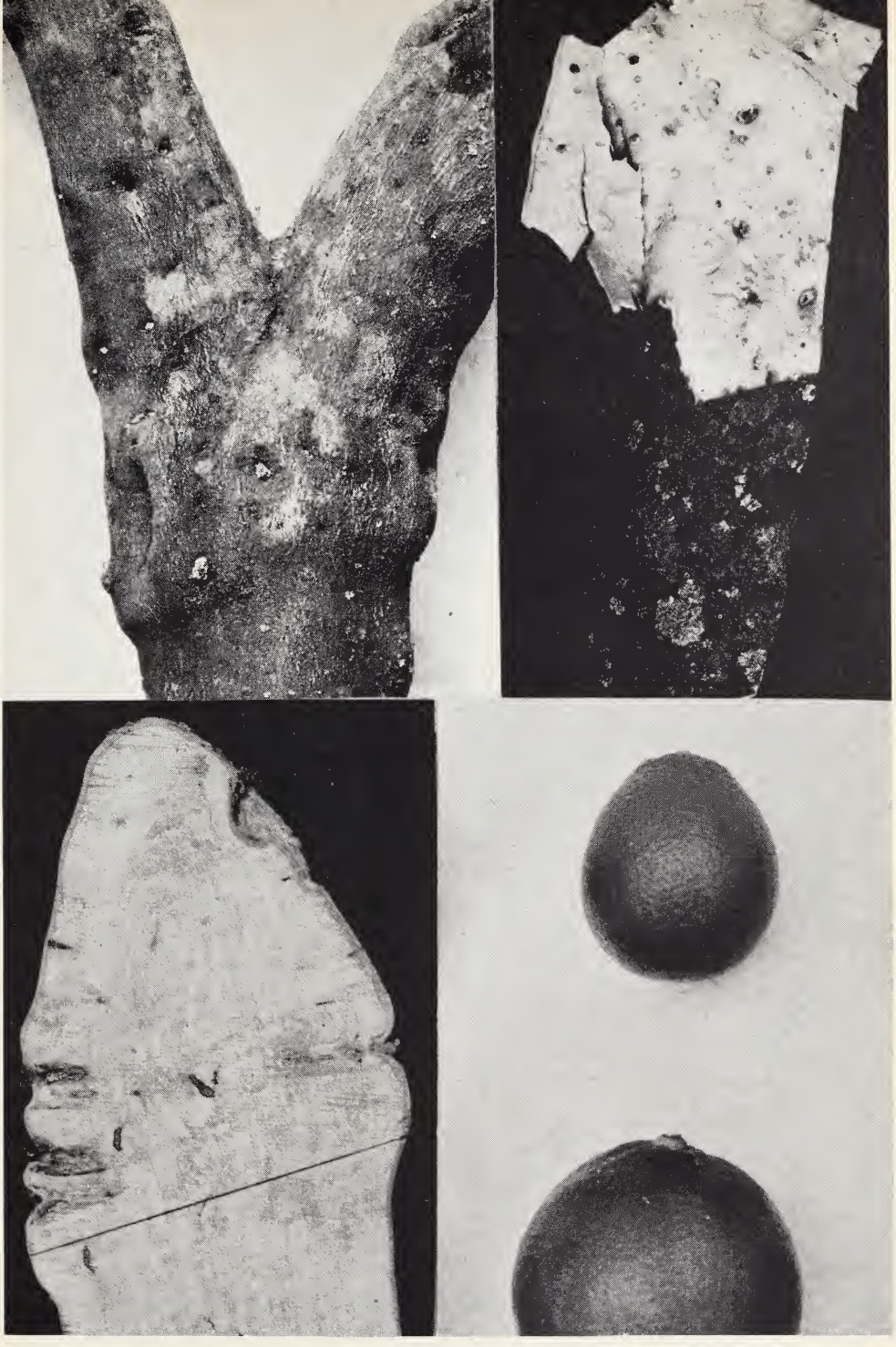


Fig. 26. Tarocco pit: (top left) surface view of affected Tarocco scion showing pits and extruded, corklike material; (top right) cambial surface with bark pulled back, exposing wood; (bottom left) longisection, showing depth of pits; (bottom right) small fruit from diseased tree and larger fruit from healthy tree.

77 per cent. Incidences of 75 per cent have been reported in Turkish lemon plantings (*20*).

The Florida investigations into possible causes of the trouble included studies on the effects of insects, mites, bacteria, water relations, heredity, fertilizers, and viruses. Much of the research was inconclusive, but several observations appear significant. Large-sized fruits developed higher incidences of rumple than did small fruits. Trees with a low incidence of rumple continued the low incidence year after year, and trees with a high incidence continued to bear large numbers of affected fruits in succeeding crops. This may suggest a connection with virus infection or the trees' genetic makeup. Trees receiving large amounts of nitrogen fertilizers had more rumpled fruits than did those given medium or low amounts. Trees with a low potassium content in their leaves showed high percentages of the disease.

With the present lack of knowledge of the cause of rumple no recommendations can be offered beyond the careful selection of budwood for propagation from disease-free trees, and judicious fertilization of the orchard.

TAROCCO PIT

On many Tarocco orange trees in the Lentini district of Sicily an unusual form of bark and wood disease, first noticed in 1962 and tentatively called Tarocco pit, has appeared (*58*).

The Tarocco scion near the bud union is most severely affected, showing large, distorted overgrowths and infoldings (fig. 26). A circular hole, ranging in diameter from 1 mm to 2cm, is found in the center of each concavity. From these holes a material like ground cork is extruded. When the bark is removed, many of these circular holes, 2 to 8 mm in diameter, are found, that have not broken through to the external bark surface. At this stage of development they are filled with a soft, whitish, meristematic tissue which later darkens as it becomes infiltrated with gum, and still later, as the lesion breaks through to the outer bark surface, produces the ground corklike material.

Buds from affected trees propagated on rootstocks of sour orange, Cleopatra mandarin, and Troyer citrange showed typical pits in the wood at the end of two to three years, but none had broken through the bark in that time.

In common with ordinary concave gum disease, the young leaves of spring growth of Tarocco trees affected by Tarocco pit show typical oak-leaf patterns and some flecking. The autumn flush of growth shows less of the oak-leaf effect and more flecks. The flecks produced in autumn are broader than those appearing in the spring. Cross-protection tests with psorosis have not been tried.

It has been possible in some instances to trace the source of the affected budwood, in others not. Sweet orange seedlings graft-inoculated with material from affected trees showed both oak-leaf patterns and flecking. Tests on Mexican lime seedlings showed no tristeza infection. Palestine sweet lime seedlings budded in 1962 with buds from trees with Tarocco pit have not yet shown cachexia-xyloporosis symptoms.

Affected trees are a third smaller than unaffected trees or trees affected by concave gum disease. Diseased trees have shorter internodes, smaller leaves, and more numerous and smaller fruits than do healthy trees, indicating the girdling effect of the trouble. The affected fruits have a more spongelike albedo, a much greater percentage of acid, and a lower solids-acid ratio than normal fruits. The ever increasing number of Tarocco orange trees found to be diseased points up the severity of the trouble on this orange variety.

WOOD POCKET

This disease, also called lignocortosis, affects certain varieties of lemon and

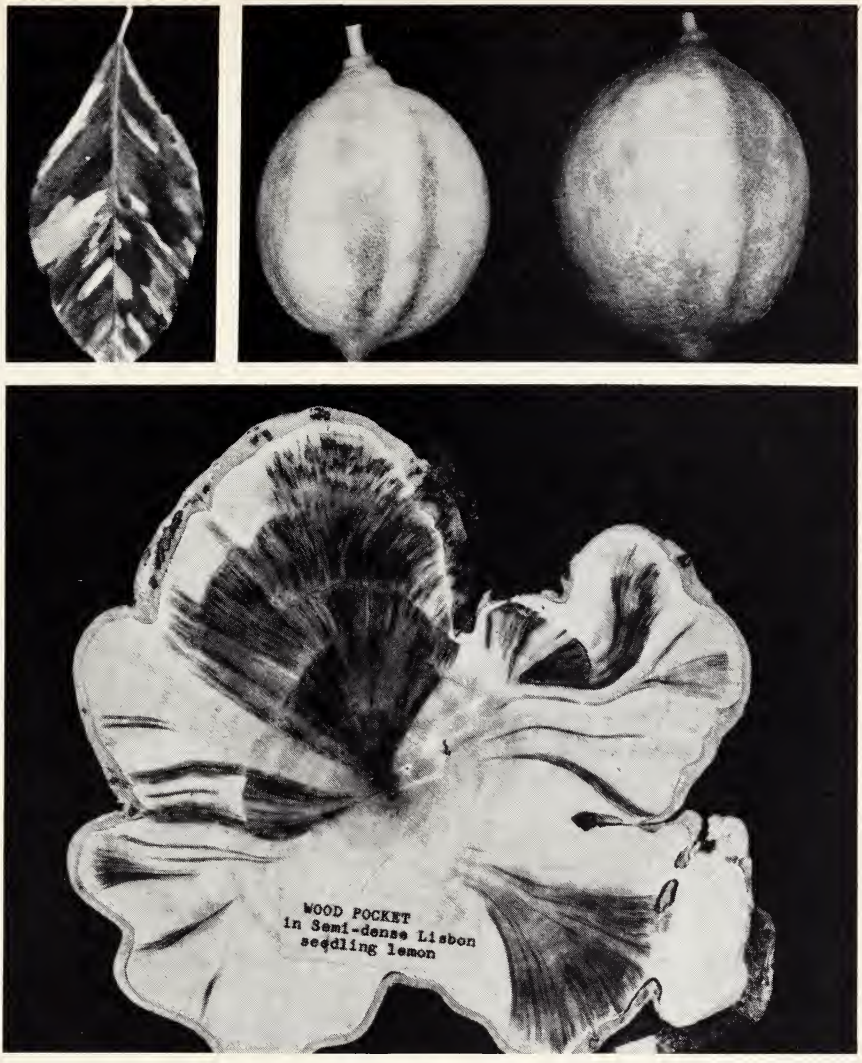


Fig. 27A. Wood pocket of Lisbon lemon: (top) symptoms on leaf and fruit; (bottom) section through trunk showing symptoms in bark and wood.

Fig. 27B. Limb of Lisbon lemon affected with wood pocket.

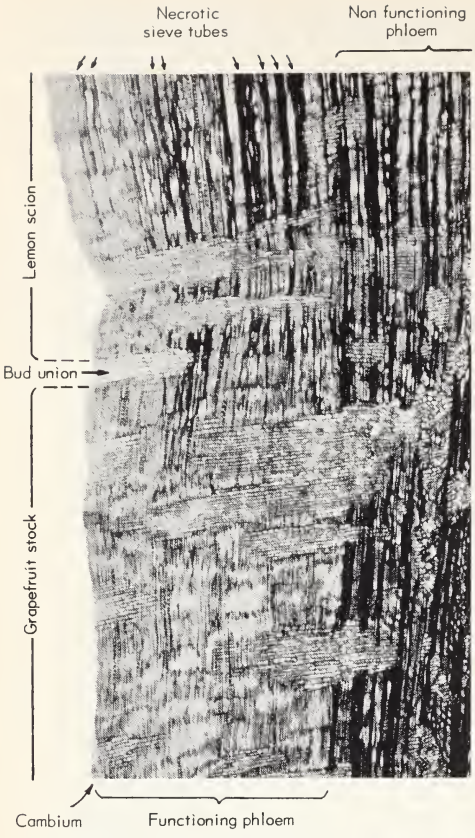


Fig. 28. Lemon sieve-tube necrosis in radial section of bark. (Photo by H. Schneider.)

lime. In California it occurs principally in the semidense Lisbon lemon (*7*). In Florida it is known also as blotch, and occurs mostly in Tahiti lime trees (*39*). The first evidence of the disease is generally a chimeralike variegation or blotching of the leaves (fig. 27A). Frequently there is considerable chimeral striping of the fruit, especially of limes. The leaf color varies from only a slight loss of green to greenish-yellow to nearly pure yellow with diverse outlines. The disease apparently is a chimeral disorder which is seed- and bud-perpetuated but which is not transmissible by grafting. A short, narrow, irregular break in the bark of the trunk or limb is the first symptom on those parts (fig. 27B). The wood underneath discolors in definite regions even before this bark symptom appears. This led to the name wood pocket or lignocortosis. Elongated areas of dead, fissured bark, 1 inch to several feet in length, form on one side of the branches or on portions of the trunk. Wood underneath becomes irregularly discolored and darkly dotted in longitudinal view. Branches lose their leaves and die back gradually, or the leaves wilt suddenly as the branches die.

LEMON SIEVE-TUBE NECROSIS

Lemon sieve-tube necrosis (fig. 28) is probably the most important of the noninfectious diseases of lemons (*52*, *65*). Eureka lemons and certain clones of other varieties are subject to the trouble; some clones are not, and do not develop it when budded with an affected clone. The older sieve tubes of three- or four-year-old trees die. During the next several years, more and more of these food-conducting vessels die, including even the youngest. Because elaborated food cannot then be transported to the roots, they starve and some die. Affected trees that had been making good growth suddenly decline and may die. In a vertical section through the phloem of lemon bark, affected sieve tubes appear as elongated, darkened areas under the microscope.

Other significant disorders of unknown cause include: Crinkle scurf of Valencia orange (*37*); a seed-perpetuated disorder of trifoliate orange resembling exocortis; sour orange rootstock necrosis (*64*), which develops when this stock is budded with Eureka lemon or with some Lisbon lemon clones; degeneration of bud union tissues when Eureka lemon is budded on trifoliate orange or Troyer citrange (*79*).

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